1087 CONTROL OF AZOTEMIC HYPERPARATHYROIDISM BY PARATHYROID AUTOTRANSPLANTATION AND 1,25-DIHYDROXYCHOLECALCIFEROL (1,25-DHCC) Ronald J. Kallen and Caldwell Esselstyn. (Spon. by Richard E. Behrman), Depts. of Pediatrics and Surgery, Cleveland Clinic, Cleveland, OH.

A 10-year-old male with congenital bilateral renal hypoplasia, chronic renal failure, and secondary hyperparathyroidism, developed bone pain and inability to walk. He was azotemic (BUN, 117 mg/dl) and had characteristic blood biochemical alterations (serum calcium, 6.5 mg/dl, phosphorus, 8.4 mg/dl, and alkaline phosphatase, 1215 units). Immunoreactive serum parathyroid hormone (iPTH) was markedly elevated, 1900 µlEq/ml (normal range, undetectable to 40 µlEq/ml). Four months later a total parathyroidectomy was done with autotransplant of a 1/8 remnant of parathyroid tissue into a sternocleidomastoid muscle. The immediate postoperative period was attended by hypocalcemia requiring sustained intravenous infusion of calcium. The iPTH declined to 520 µlEq/ml on the first postop day (preop level, 1500 µlEq/ml) and to 130 µlEq/ml on third postop day. Subsequently iPTH oscillated between 80 and 130 µlEq/ml. Two months postoperatively, hemodialysis was begun. At this time iPTH was still elevated (110 µlEq/ml) and treatment with 1,25-DHCC, 0.5 mcg/day was begun. Muscle strength improved dramatically accompanied by ability to walk. Radiologically evident bone lesions regressed and iPTH further declined to less than 40 µlEq/ml and alkaline phosphatase returned to normal. We conclude that 1,25-DHCC adequately suppressed the parathyroid remnant, by promoting adequate intestinal absorption of calcium, preventing re-emergence of secondary hyperparathyroidism.

1088 HYPERRENINEMIC ACUTE RENAL INSUFFICIENCY AND HYPERTENSION IN MINIMAL-LESION NEPHROTIC SYNDROME Ronald J. Kallen, Yuet Mei Ooi, and Kenneth Calabrese (Spon. by William Michener), Depts. of Pediatrics and Hypertension-Nephrology, Cleveland Clinic, Cleveland, OH.

A 16-year-old girl with recent onset of idiopathic nephrotic syndrome (INS) developed severe hypertension and sustained acute renal insufficiency (ARI) despite minimal lesion histology, treatment with albumin infusions, and absence of renal vein thrombosis. The nephrotic syndrome failed to respond to oral prednisone administration, "pulse" methylprednisolone, and cytotoxic agents. Peak BUN was 128 mg/dl; peak serum creatinine, 5.5 mg/dl. Plasma renin activity (PRA) was 49 ng/ml/hr. Plasma volume was 70% of normal. Elevated PRA was partially suppressed by albumin infusion. Azotemia persisted for 3 months. BUN declined to 18 mg/dl and serum creatinine to 1.8 mg/dl concurrent with tapering of prednisone dosage. We speculate that this patient had incipient vasomotor nephropathy due to angiotensin-mediated renal vasoconstriction consequent to multiple factors: 1. corticosteroid-induced vascular sensitization to angiotensin (Am. J. Med. 58:216,1975); 2. hypovolemia; and 3. treatment with renin-raising drugs (hydralazine, furosemide). We suggest that patients with INS and ARI may benefit from treatment with an angiotensin-antagonist.

1089 CLASSIFICATION OF HEMOLYTIC UREMIC SYNDROME(HUS).Bernard S.Kaplan and Jean-Pierre de Chadarevian.McGill Univ.-Montreal Children's Hosp.Research Inst.,Depts. of Nephrology and Pathology,Montreal,Canada.

HUS is not a discrete clinicopathologic entity but a syndrome conceptually akin to the nephrotic syndrome.Based on our studies we present this classification:

1)The typical,idiopathic HUS of infancy and childhood:Commonest form.Mainly in endemic areas,but also sporadic.Usually < 4 yrs. Prognosis:mild cases,excellent;severe,very good.If sib affected, occurs within days.Pathology,electron microscopy(EM):endothelial cell swelling and formation of subendothelial space.Thromboses rare. 2)HUS with a possible genetic predisposition:Sibs with onsets > 1 yr apart.Mainly nonendemic areas.Poor prognosis with inexorable course.EM:no apparent changes to severe damage.Possibly some overlap with Group 3.May be inherited in some cases as an autosomal dominant gene. 3)HUS with recurrent episodes:Data on 29 cases,24 from nonendemic areas.Rarely have typical prodrome.Only 50% < 4 yr at onset.Sib or cousin involvement in 27%;prognosis poor, > 30% die.EM pathology ranges from no apparent lesion to severe glomerular injury. 4)HUS associated with pregnancy:Pre-eclamptic toxemia,postpartum renal failure. 5)HUS with a putative etiology:Oral contraceptives,viruses,Shigella,Salmonella. 6)Atypical HUS:Classical clinical findings but subepithelial deposits on EM and no subendothelial lesions. 7)HUS as a feature of other syndromes:Malignant hypertension,TTP,malignancy,immunodeficiency syndromes,postrenal transplantation.

1090 Na REABSORPTION AND K SECRETION IN THE DISTAL TUBULE OF NEWBORN DOGS. L. I. Kleinman & R.O. Banks, Departments of Pediatrics & Physiology, University of Cincinnati, College of Medicine, Cincinnati, Ohio.

Newborns do not excrete infused saline loads as efficiently as do adults. Proximal tubular Na reabsorption is similar in newborn and adult dogs under control and saline expansion (SE) conditions (A.J.P. 228:1403, 1975). Differences occur in more distal segments of the nephron. In order to evaluate Na reabsorption in the distal convoluted and cortical collecting tubule, Na reabsorption was measured in 26 newborn and 9 adult dogs before and during maximal inhibition with amiloride, a drug known to inhibit Na reabsorption and K secretion in these regions. Under control conditions 3.6±.3% of the filtered Na was reabsorbed in the amiloride sensitive region of the nephron (ASR) in the puppy compared to 2.3±.4% for the adult ($p<.05$). Without amiloride, SE increased total fractional Na excretion 8.0±2.3% in adults and 1.9±.4% in puppies. When the ASR was inhibited SE increased fractional Na excretion 7.2±1.0% in adults and 4.5±1.2% in puppies. During SE, fractional Na reabsorption in the ASR was higher ($p<.05$) in the puppy (5.3±.4%) than in the adult (2.8±1.2%). K secretion in the ASR was the same in the puppy (1.4±.2 meq/ml GFR) and the adult (1.4±.1) during control as well as during SE, (1.4±.2 for puppies and 1.5±.4 for adults). There was no correlation between Na reabsorbed and K secreted in the ASR for both puppies and adults during control or SE periods. These results support the conclusion that the ASR contributes to some but not all of the attenuated response of the newborn to saline expansion. In addition,Na reabsorption and K secretion in this region are dissociated.

1091 MATURATION OF RENAL ENZYMES IN NEWBORN RATS. L. I. Kleinman, H. Wald, J.W. Czazkes, Dept. of Pediatrics, University of Cincinnati Medical School, Cincinnati, Ohio and Hadassah Hospital, Jerusalem, Israel.

Adenyl cyclase (AC), an enzyme involved in cyclic AMP synthesis, phosphodiesterase (PDE), involved in cyclic AMP breakdown, and Na-K-activated ATPase, involved in Na reabsorption, were measured in 37 groups of rats 3 to 6 weeks and adults, in renal cortex, medulla, and papilla. There was a gradient of AC activity from papilla to cortex in all age groups* but this gradient was less in the newborn animals*. In cortex and medulla, AC was highest (by 25%)* in 3 week rats and reached adult levels by 6 weeks. In papilla, AC was 70% of adult values* at 3 weeks and rose to 80% at 6 weeks*. In cortex, PDE was highest in the most immature rats and decreased with maturation*. In papilla, PDE was 50% higher* in 3 week rats than adults but by 6 weeks the enzyme level fell to those of the adult. The low AC and high PDE activities in papilla of newborn rats suggest a low net cyclic AMP production there. ATPase activities were highest in medulla and lowest in papilla of all animals*. Medullary ATPase was only 64%* of that of adults at 3 weeks and rose to 73%* at 6 weeks. There were no significant maturational changes in cortical and papillary ATPase. When placed on a high Na diet, newborn animals increased cortical ATPase 30%* and medullary ATPase 100%* over controls. These results demonstrate that maturation of renal enzymes vary in different regions of the kidney and are related to the specific functions of and stimuli to these regions. Maturational characteristics can be modified by altering the stimulus. *p<.01

1092 EFFECT OF POLYCYTHEMIA ON RENAL FUNCTION IN NEWBORN DOGS. Uma R. Kotagal, Theresa Disney, Leonard L Kleinman, University of Cincinnati College of Medicine, Dept. of Pediatrics, Cincinnati, Ohio 45267 USA

The effect of increasing Hct on renal hemodynamics and function was studied in 12 newborn dogs 1-20 days old. When the puppies were made polycythemic by exchange transfusion with packed RBC's to raise the Hct from 32.9±2.5 to 65.6±1.3**, cardiac output ($ml.Kg^{-1}min.^{-1}$) fell from 217.3±17.8 to 122.9±12.0**, mean B.P (mmHg) increased from 73.8±6.9 to 95.5±2.6** and peripheral vascular resistance ($mmHg.ml.^{-1}kg.^{-1}min.^{-1}$) increased from .40± .04 to .95±.11*. The percent of cardiac output distributed to the kidney increased from 6.6±.7 to 10.5±.69**. Renal blood flow was not significantly altered but renal vascular resistance ($mmHg.ml.^{-1}min.^{-1}$) increased from 63.9±3.4 to 72.2±2.9* and renal plasma flow ($ml.g^{-1}min.^{-1}$) decreased from .87±.07 to .46±.02**. Intrarenal blood flow distribution was altered in that the ratio of flow to the inner cortex divided by outer cortex (IC/OC) increased from .33±.03 to .51±.03**. This was due primarily to an increase in IC flow ($ml.min.^{-1}gIC^{-1}$) from 7.3±.8 to 9.9±.9**. The decrease in OC flow ($ml.min.^{-1}gOC^{-1}$) from 23.2±4.4 to 19.9±1.9 was not significant. GFR ($ml.min.^{-1}g^{-1}$) decreased from .491±.14 to .158±.05**. Na reabsorption ($\mu eq.min.^{-1}ml.GFR^{-1}$) decreased from 122.4±3.9 to 116.6±4.41* and K reabsorption ($\mu eq.min.^{-1}ml.GFR^{-1}$) decreased from 4.37±.39 to 2.92±.64*. Thus, polycythemia depresses cardiac output, increases peripheral vascular resistance, decreases renal plasma flow, alters intrarenal blood flow distribution and affects both glomerular and tubular function.

*p<.05 **p<.01